IDCases 19 (2020) e00679

Contents lists available at ScienceDirect

IDCases

journal homepage: www.elsevier.com/locate/idcr

Case illustrated

A budding case of infectious endocarditis: *Candida lusitaniae*

Elham Rahmati[a,*,1], Adrian J. Correa[b], Rosemary C. She[b]

[a] Department of Medicine, Division of Infectious Diseases, Keck School of Medicine of the University of Southern California, Los Angeles, CA, United States
[b] Department of Pathology, Keck School of Medicine of the University of Southern California, Los Angeles, CA, United States

ARTICLE INFO

Article history:
Received 31 October 2019
Received in revised form 5 December 2019
Accepted 6 December 2019

Keywords:
Candida lusitaniae
Infective endocarditis
Fungal endocarditis
Amphotericin resistance
Antifungal resistance

ABSTRACT

This is a case of recurrent *Candida lusitaniae* prosthetic valve endocarditis with budding yeast and pseudohyphae on the histopathology. This case illustrates the importance of keeping vigilant in recognizing some of the emerging drug resistant Candida species in our practice.

Case

A 41-year-old Caucasian female with past medical history of intravenous drug abuse, chronic hepatitis C, and bioprosthetic mitral valve replacement due to infectious endocarditis was transferred to our facility for persistent fungemia and recurrent endocarditis. The original mitral valve replacement, presumptively due to a fungal organism, dated back approximately two years prior to presentation. Her initial presentation was also complicated by embolic events including thrombosis of the superficial femoral artery which required left above the knee amputation. She had a subsequent admission for thrombosis of the celiac artery and *Candida lusitaniae* fungemia. She was discharged with plans for long term anticoagulation as well as 8 weeks course of intravenous micafungin. However, shortly after she was readmitted to an outside facility for left axillary artery thrombosis. During her stay *C. lusitaniae* was isolated in 5 sets of blood cultures despite continued treatment with micafungin. Two weeks into admission, a transesophageal echocardiogram demonstrated mitral valve vegetation. The patient was transferred to our facility for valve replacement.

On evaluation at our facility, she was afebrile (36.9 °C), normotensive, but tachycardic to 138 beats per minute. She was non-toxic appearing and no murmurs were appreciated on exam. Her left upper extremity was erythematous, warm to touch, and tender to palpation. Although her left hand was non-cyanotic with good capillary refill and intact sensation, brachial and radial pulses were not easily palpable. She had an elevated white blood cell count of 17.7×10^9/L, hemoglobin of 11.8 g/dL and elevated platelet count of 487×10^9/L. Computed tomography angiogram of the left arm revealed an abrupt cut-off of the left axillary artery consistent with occlusive thrombosis. Two additional sets of blood cultures recovered *C. lusitaniae* while the patient was on micafungin therapy. Three days after transfer, therapy was changed to fluconazole but *C. lusitaniae* detected again in blood cultures drawn 2 days later. At this time the patient underwent redo mitral valve replacement. *C. lusitaniae* was recovered from the resected valve tissue. Blood cultures were negative in 5 sets drawn post-operatively. All identifications were performed by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry. Budding yeast and pseudohyphae on histopathology also confirmed infective endocarditis of the prosthetic valve by this organism (Fig. 1). Recurrence was thought to be due to high cloth burden in the celiac artery which was not amenable to embolectomy by interventional radiology. Subsequently, the patient underwent left arm embolectomy. *In vitro* susceptibility testing of the organisms revealed minimal inhibitory concentrations (MIC) of $\leq$ 1 μg/mL to fluconazole, 0.5 μg/mL to caspofungin, and $\leq$ 0.12 μg/mL to voriconazole. There are no interpretive breakpoints according to current Clinical and Laboratory Standards Institute (CLSI) M60 guidelines. Given the intrinsic resistance of *C. lusitaniae* to amphotericin B, reporting of MIC is not recommended by CLSI, as even low amphotericin B MIC have not shown to be predictive of positive clinical outcomes. After initial therapy with micafungin, the patient was successfully treated with

* Corresponding author at: Department of Infectious Diseases, University of California San Francisco, Fresno 155 N. Fresno Street #307, Fresno, CA 93701, United States.
E-mail address: erahmati@fresno.ucsf.edu (E. Rahmati).
[1] Current Affiliation: Division of Infectious Diseases, University of California San Francisco, Fresno, CA

https://doi.org/10.1016/j.idcr.2019.e00679

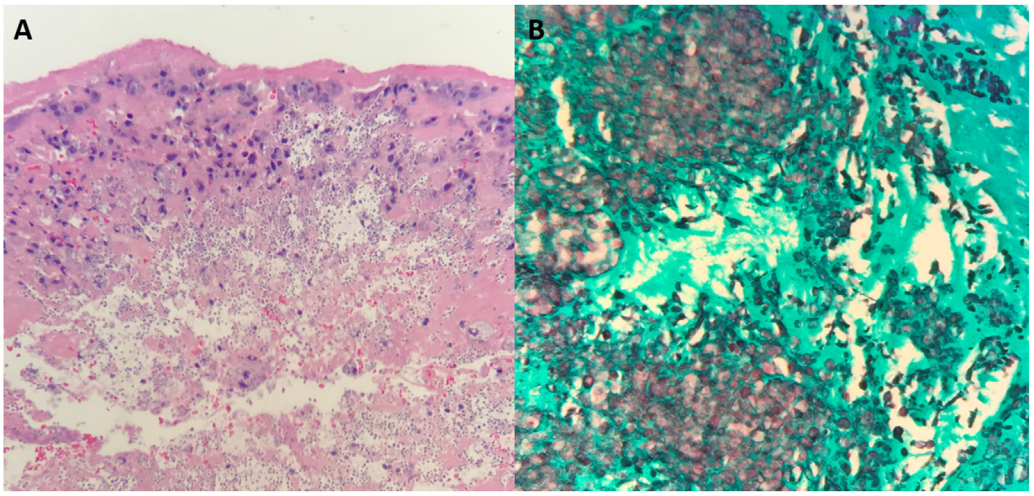

Fig. 1. A. Hematoxylin and eosin-stained mitral valve tissue section shows infiltration by faintly staining yeast cells (200X magnification). B. Gomori methenamine silver stain highlights numerous budding yeast and occasional pseudohyphal forms in the mitral valve corresponding to *C. lusitaniae* recovered from culture of the valve tissue and blood (400X magnification).

fluconazole 800 mg once a day for 6 weeks with plans to transition to lower dose oral fluconazole for one year. The patient was lost to follow-up.

Discussion

Although *Candida* species are the most frequent fungal cause of infective endocarditis, *C. lusitaniae* is a rare etiology of the disease. Only a handful of cases have been documented in the literature with the first reported case of prosthetic valve endocarditis described in 1998 [1,2]. *C. lusitaniae* is notable for its resistance to amphotericin B, initially described in 1979. [3] Infections by this species respond poorly to amphotericin in spite of *in vitro* testing frequently indicating low MICs. [3,4] Whole genome sequencing has identified genetic similarities between *C. lusitaniae* and the recently emerging *C. auris* which can demonstrate resistance to multiple antifungal agents. [5] In conclusion, in treatment of *C. lusitaniae*, it is crucial to recognize its resistance to amphotericin B and that first-line azole therapy frequently results in good outcomes in conjunction with surgical interventions.

Funding

Not applicable.

Consent

Not applicable.

Author contribution

All authors have contributed to the work.

Declaration of Competing Interest

The authors declare that there are no conflicts of interest.

References

[1] Wendt B, Hagland L, Razavi A, Rath R. *Candida lusitaniae*: an uncommon cause of prosthetic valve endocarditis. Clin Infect Dis 1998;26:769–70.
[2] Michel RG, Kinasewitz GT, Drevets DA, Levin JH, Warden DW. Prosthetic valve endocarditis caused by *Candida lusitaniae*, an uncommon pathogen: a case report. J Med Case Rep 2009;3:7611.
[3] Pappagianis D, Collins MS, Hector R, Remington J. Development of resistance to amphotericin B in *Candida lusitaniae* infecting a human. Antimicrob Agents Chemother 1979;16:123–6.
[4] Hawkins JM, Baddour LM. *Candida lusitaniae* infections in the era of fluconazole availability. Clin Infect Dis 2003;36:e14–8.
[5] Sharma C, Kumar N, Pandey R, Meis JF, Chowdhary A. Whole genome sequencing of emerging multidrug resistant *Candida auris* isolates in India demonstrates low genetic variation. New Microbes New Infect 2016;13:77–82.